FEATURES OF CYTOKINE STATUS IN PATIENTS WITH COMORBIDITY OF COPD AND CHRONIC PANCREATITIS

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The purpose. The aim of this study was to evaluate the status of serum levels of interleukin-1β (IL-1β) and tumor necrosis factor alpha (TNF-α), in patients with concomitant course of COPD and chronic pancreatitis.

Materials and methods. With this goal were examined 59 individuals (32 patients with COPD. In combination with chronic pancreatitis in a phase of unstable remission, and 27 - with an isolated course of COPD II-III century.). Diagnosis is established on the basis of clinical and anamnestic data, laboratory data and instrumental methods. To determine the regulatory indicators were also surveyed a group of healthy individuals (20 people). The level of proinflammatory cytokines was evaluated by determining the content of pro-inflammatory cytokines: interleukin-1β and tumor necrosis factor - alpha - a standard set of "Protein contour", St. Petersburg. The results were processed using statistical programs.
The results. The study showed that in the group with COPD over an isolated value of TNF-α was 38,3 ± 3,7 ng / l, and IL-1β - 42,9 ± 4,1 ng / l, 1.6 and 1 and 7 times respectively higher than in controls (P <0.001). In determining the levels of proinflammatory cytokines in patients with concomitant COPD course of chronic pancreatitis and TNF-α level was 92,3 ± 4,3 ng / l, and IL-1β - 72,3 ± 3,8 ng / L, which exceeded the performance standards 3.8-and 2.8-fold, respectively (P <0.001).
Conclusions. Exacerbation of COPD was accompanied by a phase of significant increase in the levels of proinflammatory cytokines in patients with isolated over as COPD, and when it combined with chronic pancreatitis. And when comorbid pathologies within these levels of TNF-α and IL-1β were significantly higher than in patients with isolated over COPD. Thus, the presence of concomitant chronic pancreatitis in patients with COPD leads to a significant increase of inflammatory reactions that can be regarded as prognostically unfavorable factor.